IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
TEXARKANA DIVISION

THE STATE OF TEXAS Plaintiff VS. THE AMERICAN TOBACCO COMPANY; R.J. REYNOLDS TOBACCO COMPANY; BROWN & WILLIAMSON TOBACCO CORPORATION; B.A.T. INDUSTRIES, P.L.C.; PHILIP MORRIS, INC.; LIGGETT GROUP, INC.; LORILLARD TOBACCO COMPANY, INC.; UNITED STATES TOBACCO COMPANY; HILL & KNOWLTON, INC.; THE COUNCIL FOR TOBACCO RESEARCH-USA, INC. (Successor to Tobacco Institute Research Committe); and THE TOBACCO INSTITUTE, INC.	: : : : : : : CIVIL ACTION : NO. 5-96CV91 : : : UNITED STATES JUDGE: : DAVID FOLSOM : : : UNITED STATES MAGISTRATE: : WENDELL C. RADFORD : : :	

DEPOSITION OF MICHAEL SPEER, M.D.

TAKEN ON SEPTEMBER 4, 1997

Called as a witness by the Defendants, taken before Linda Tate, a Certified Shorthand Reporter and Notary Public in and for the State of Texas, on the 4th day of August 1997, beginning at 8:00 a.m., at Texas Children's Hospital, Room 8340, Houston, Texas, pursuant to Notice and the Federal Rules of Civil Procedure.

A P P E A R A N C E S

Counsel for the Plaintiff:

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By: MR. MICHAEL MINTON

PROCEEDINGS

THE VIDEOGRAPHER: It's September 4th, 1997. The approximate time is 8:10 a.m. This is the continuation of the deposition from September 3rd, Dr. Speer. We're on the record.

Thereupon,

MICHAEL SPEER, M.D.,

resumed the stand, having been previously cautioned and sworn, was examined and testified further upon his oath as follows:

EXAMINATION CONTINUED

BY MR. MINTON:

Q. Good morning again, Dr. Speer.

A. Good morning.

Q. Did you have a chance to review any materials last night with respect to your deposition?

A. Are you kidding?

Q. No, I'm not.

A. No, I did not.

Q. Okay. Did you speak with anyone about your deposition?

A. No.

Q. Dr. Speer, I would like to ask you about the

portion of your opinions that deals with prematurity.

A. All right.

Q. The document that was provided to us, Exhibit 6, that contains your opinion, indicates that you have the opinion that smoking is associated with premature births, correct?

A. Correct.

Q. All right. And as I understand it, you've not made any sort of methodologic analysis regarding that association, correct?

A. Correct.

Q. All right. And that opinion regarding an association is based upon articles that you have seen from time to time working as a clinical neonatologist, correct?

A. And textbooks.

Q. And textbooks. But you -- would it be fair to say that you have not done research regarding the underlying study methodologies in studies that have reported such an association?

A. I think it's stronger than an association. I think it's a cause and effect relationship.

Q. Well, that being said, have you investigated

the methodology of the studies that have reported --

A. No, I have accepted the statements of authors that write in textbooks that are used in the field of neonatology.

Q. All right. And is that true with respect to the gamut of the opinions that are expressed in Exhibit 6, that you've not done any methodologic analysis on your own; you've accepted the opinion of others?

A. I have not done any research in this area, correct.

Q. All right. And so any -- any opinion that you're providing us in that document is a reflection of opinions of authors of other documents or textbooks?

A. And training and articles, as you noted, that I have read in the past, correct.

Q. With respect to the information that you have become aware of regarding maternal smoking and prematurity, have you seen negative studies, in other words, studies that have failed to report an association between maternal smoking and prematurity?

A. Not that I can think of off the top of my

head.
Q. All right. Would those be significant to you if they existed?
A. They might be. It depends on the methodology used in the study, the size of the sample, et cetera.
Q. All right. That is the key to any of these studies, then, an analysis of the methodology?
A. It not so much an analysis of the methodology. It is correct methods -- are correct methods used in designing and carrying out the study.
Q. All right. And if there were correctly designed studies that failed to find a statistically significant relationship between maternal smoking and prematurity, would that be of significance to you?
A. I think I just answered that.
Q. If they were large enough and well done --
A. And if you then took that analysis and looked at all of the studies that now deal with that issue, then you could probably get some sense of where the preponderance of evidence is. My opinion is that the preponderance of evidence says that smoking and low birth weight,

prematurity are related.

Q. Okay. The -- and we covered yesterday at some length the issue of causal criteria. I take it that your opinion about the relationship between maternal smoking and prematurity is a reflection of the opinions of others regarding reaching the conclusion of causation; is that correct?

A. What I'm saying is, that smoking is a cause, not the only cause by any stretch of the imagination, but a cause of prematurity and low birth weight.

Q. All right. But my question was directed more towards the types of things we were discussing yesterday in terms of what criteria scientists use in evaluating epidemiologic studies in order to make a judgment about whether a statistical association demonstrated in epidemiologic studies is likely to be causal. Do you recall that discussion?

A. And I also recall telling you that I was not an epidemiologist or a statistician.

Q. And therefore, to the extent that your opinion on causation is offered here, it is offered on the basis of a causal analysis done by other

people and not by you, correct?

A. Correct.

Q. All right. Can you tell us, Dr. Speer, in the studies that did find a statistically significant difference in the length of gestation among mothers who smoked, what the mean difference in length of gestation was?

A. No, I cannot remember that.

Q. All right. If the studies that found a change in gestation length among mothers who smoke compared to mothers who did not smoke was in the neighborhood of three to four days, would that be of clinical significance?

A. Can be.

Q. A change in gestational length of three to four days can be of clinical significance?

A. If you happen to be on the bottom of the bell shaped curve and you should have been 25 weeks and you're now 24 and a half weeks, that's not good.

Q. All right. Other than the lower left asymptote of the curve, it would not be of clinical significance, correct?

A. Depends on the patient.

Q. All right. If a baby --

A. If a patient, for example, is a baby of a diabetic mother who is born at 34 weeks as opposed to 35 weeks, there is an increased risk of hyaline membrane disease in that particular patient because the surfactant system that governs whether a patient has hyaline membrane disease, a lung disease of prematurity, kicks in later in a diabetic infant at -- that's born of a diabetic mother, I'm sorry, not a diabetic infant -- than in the normal population. So given an individual patient, there can be a significant difference in the need for medical support on a basis of three to four days.

Q. All right. And I believe --

A. It is not an absolute.

Q. All right. We believe -- I believe we established yesterday that in terms of viewing an outcome after it has occurred in a person with multiple risk factors, there is no way we can go back and assign causality, total or partial, to any particular risk factor, correct?

A. As a general, global statement, I would agree with you.

Q. All right. And with respect to the issues we've discussed in this deposition, that is also a true statement, is it not?
A. If you take the patient who already has the problem and look at it post-delivery, it is difficult to say what caused the problem, in many instances.
Q. Well, I won't re-argue the point. That was a response that you gave yesterday and then we went on to say that in fact it was clinically impossible to go back and allocate causality in that patient among the various risk factors that may have been present, correct?
A. Asked and answered.
Q. If -- have you -- have you reviewed any data regarding mean or population-based gestational age differences among mothers who smoke as compared to mothers who do not smoke?
A. I think -- are you asking once again have I done a methodological review of the literature?
Q. No. I'm just asking if you have seen gestational age data plotted out anywhere in a histogram-type form comparing the smoking group with the nonsmoking group?

A. Probably.
Q. All right. And does it look like -- you remember the exhibit that I attempted to draw yesterday which plotted out birth weights and showed two gaussian distributions, one shifted left of the other?
A. If that was the -- if you're -- if you're talking about the difference in birth weight between White and Black population?
Q. Yes.
A. Yes, I remember that.
Q. All right. And is that the same picture that you see when you compare the gestational age of smokers versus nonsmokers, that you see a uniformly distributed curve, one shifted to the left of other, and that one that's shifted left is the smoking curve?
A. The data that you're talking about, I have not reviewed in a number of years, so I cannot tell you how much that shift is.
Q. All right. So would it be fair to say that if you don't know whether or not the length of gestation is found to be uniformly shifted, whether or not statistically that negates the possibility that changes in mean birth weight

between the groups are due to extreme effects in a few cases?

A. I think what you just said, to put it in English, is that I -- if -- that there is a shift in the curve because of bias of a few patients. My understanding of the data is that that statement is erroneous that you just made and that smoking is associated causally -- a cause for prematurity. Period. I think that's the simplest way to look at it.

Q. What I was attempting to ask is: Since you don't know what the nature of the shift in the curve is in terms of length of gestation, can you tell us whether or not the mean length of gestation difference that has been found is due to uniform changes among all of the point constituents on the curve or among a few extreme effects?

A. That is my understanding.

Q. The former or the latter?

A. The former.

Q. That there is a uniform difference among all points along the curve?

A. There is a difference between the populations. I don't know whether it's every

single point along the curve, but the population of babies born to mothers who smoke are more premature than babies born of mothers who don't smoke.

Q. All right. But the point you just added there was that is your belief or your opinion that the reason that is so is not because of a few extreme effects that impact that shift in the mean, but because of a uniform distributed change, correct?

A. That's my understanding.

Q. All right. Do you know if the Surgeon General has said that maternal smoking does not appear to have much effect on mean gestational age?

A. I don't remember that.

Q. All right. Is that a statement with which you agree?

A. I'm sorry?

Q. That maternal smoking does not appear to have much effect on mean gestational age.

A. I think we just spent the last ten minutes getting the opinion that I have that it does have an effect on gestational age.

Q. Well, what I'm trying to do is zero in on any information you have on the change in the mean

value. And I -- is the answer you simply don't know what the change --

A. No, I've already answered that question.

Q. All right. What is the change in the mean? Is it --

A. I've said babies born of mothers who smoke are more premature as a group than babies born of mothers who don't smoke.

Q. Six hours more premature? Five days more premature? How much?

A. A significantly different gestational age in the two groups. Now, I've already told you, I don't know exactly where the mean is and I don't know exactly what the numbers are. You implied four to five days and it sounded like you were speaking from some knowledge that you had recently reviewed. Fine. The fact of the matter is, they are more premature.

Q. But how much more premature, you're not prepared to say?

A. Anything that makes you more premature is not a good idea.

Q. Well, is -- six hours isn't going to have any clinical significance, is it?

A. I don't know.

Q. Okay.
A. Depends on the baby. I imagine you could probably find someone where six hours did make a difference. In general, no, it doesn't make a difference.
Q. Are you aware of any studies that have associated maternal smoking with extreme prematurity or very premature babies?
A. There may well be studies out there, but if you and I both agree that all of the curves are shifted, then that means that you're going to shift the more premature babies and the less premature babies at any given gestational age.
Q. So that would -- that would tend --
A. So that would be a yes.
Q. Okay. What are the most prevalent complications leading to preterm delivery?
A. I'm sorry?
Q. What are the most prevalent complications of pregnancy leading to premature delivery?
A. Well, not being an obstetrician, I probably can't give you as full an answer as you could get from my obstetrical colleagues. I don't know which is the most common cause of

prematurity. I can tell you there are many causes of prematurity which you implied.

Certainly we see prematurity in mothers who have undergrown fetuses that are not growing in utero and it is deemed important to deliver that baby in a premature state as opposed to a nonpremature state for the welfare of the baby and sometimes the welfare of the mother.

Q. That would be called iatrogenic prematurity?

A. No, that's because you have a baby that's small-for-dates in utero that's not growing and you deliver them because of placental insufficiency or whatever the other causes are that the baby is not growing.

Q. All right. That -- as far as you know, that particular classification is not known as iatrogenic prematurity?

A. We many times know why the baby is small, so he's not iatrogenic. We're doing it on purpose.

Q. No, I mean -- well, I didn't mean idiopathic, I meant iatrogenic.

A. I don't think so. I've never heard that term used in that fashion.

Q. Okay. I'm sorry. I interrupted you. You were talking about other causes of prematurity.

A. Some moms go into labor, they have incompetent cervices and they go into labor prematurely. Sometimes it is in response to medication use on the part of the mother, either elicit, legal or un -- or not legal.

Sometimes babies are delivered prematurely because there is an occult or nonoccult infection of the vaginal tract involving the fetal membranes stimulating labor. I think we talked about this briefly yesterday.

Q. Okay. While we're on the topic of occult or nonoccult infections, is chorioamnionitis one of those nonoccult infections?

A. It can be occult or nonoccult.

Q. All right. And is that a relatively prevalent condition?

A. By what definition do you wish to use?

Q. Well --

A. Because there are several, or at least two.

Q. Can you explain what the difference is between the two?

A. One's clinical and one's pathological, without clinical findings.
Q. All right. Would it be fair to say that there is a lot of chorioamnionitis that is missed?
A. Again, what definition are you wishing to use? If you're using a clinical definition, the answer is no. If you're using only a pathologic definition that may have no clinical correlate and may have no effect on the baby, yes.
Q. All right. Is there -- is there chorioamnionitis that can only be detected by an inspection of the placenta?
A. Correct.
Q. And are you suggesting that if the -- if the signs or symptoms of chorioamnionitis are not sufficient to be clinically frank in terms of being apparent without an inspection of the placenta, that they are insufficient to cause any form of fetal injury?
A. That's not what I said.
Q. Okay. That -- it is clear that stages of chorioamnionitis that are not sufficiently frank to be detected by any means other than by inspection of the placenta are nonetheless

capable of producing fetal effects, adverse fetal effects, correct?

A. That's a hypothesis that has not been proven.

Q. Is that a hypothesis that you have investigated?

A. If by "investigated" you mean have I researched -- done research in the area, no, I have not done research in the area.

Q. I'm going to try to briefly go through a list of conditions, Dr. Speer, and get your opinion on whether or not these are risk factors that are associated with the adverse pregnancy outcomes that you have noted in Exhibit 6. All right?

Socioeconomic studies?

A. For all of the issues that I raised in Exhibit 6?

Q. Yes.

A. We're just sort of going to blanket say socioeconomic status is responsible or has an influence on all of them?

Q. Yes.

A. No, it does not have an influence on all of them. It has an influence on some of them.

Q. All right. And which of the conditions that

you've identified has socioeconomic status not been associated with?

A. I don't think there is a socioeconomic status -- well, there may be in certain populations. You're asking a global question that's very difficult to answer without --

Q. Because the impact of socioeconomic status may be different from population to population?

A. Yes.

Q. All right. And so in order to determine the effect of socioeconomic status, we have to look at a particular population?

A. I think that would be a fair assessment.

Q. And if we are looking at a population of low socioeconomic status, are there conditions about which you have provided opinions in Exhibit 6 that seem to not be associated with socioeconomic status?

A. Again, if you -- it depends on the population you're dealing with. If you're saying socioeconomic status alone, there are issues here, particularly those that have to do with nicotine and carbon monoxide, that if you're a low socioeconomic status person and don't smoke, you don't have those risk factors.

If you are a low socioeconomic person who does smoke, then you have those risk factors, for example. But is that due -- is that truly due to being low socioeconomic status or is that due to other factors --

Q. In other words --

A. -- as to why they smoke?

Q. -- what you're saying is smoking is linked to socioeconomic status?

A. I'm saying that although people in lower educated groups of populations have a higher incidence of smoking, is it truly due to their socioeconomic status that they have a higher incidence of smoking or is it due to some other parameter such as peer pressure and has nothing to do with socioeconomic status and you have true, true and unrelated.

Q. Well, you are suggesting that smoking is linked to socioeconomic status?

A. No, I'm suggesting that they may be two independent variables and smoking occurs more often in people of low socioeconomic status, but it's not due to their low socioeconomic status that they smoke.

Q. Okay. Is socioeconomic status recognized to

be a risk factor apart from its potential confounding impact with respect to each of the health effects that you've listed?

A. Socioeconomic status in and by itself, now, does have a relationship with prematurity, reduced birth weight and other morbidities that are listed in Exhibit 6.

I am not sure -- and once again, you'd have to correct now for intellectual prowess in regard to the mental retardation issue. Just because you are poor doesn't mean you're stupid or ignorant, so for as your intellectual abilities to learn.

However, if you are not intelligent enough to learn, then certainly you are mentally retarded, by definition. So again, you have to be very clear in the definitions to what you're trying to say.

Q. All right. So -- but with respect to spontaneous abortions, reduced birth weight, premature birth, abruptio placenta and placental injury, socio --

A. I didn't say placental injury and I don't think I said abruptio placenta.

Q. Okay.

A. I said prematurity and low birth weight.
Q. How about spontaneous abortions?
A. I think that's true.
Q. All right. How about SIDS?
A. Once again, you have an increased risk of SIDS in population groups that commonly are associated with a lower socioeconomic status. However, you have the information that SIDS appears to be much more prevalent in certain racial groups, such as American Indians or Black, regardless of their socioeconomic status.
Q. Okay. So there is an ethnic association as well?
A. Correct.
Q. Based on what you told us yesterday, is it clear or is it true that you are unable to rank socioeconomic status with respect to its important as -- its importance -- I'm going to start over.

Is it true that you're unable to rank socioeconomic status as a risk factor against maternal smoking in terms of its relative impact in producing spontaneous abortions, reduced birth weight, premature

births?

A. I'm not too sure I said that.

Q. All right. Are you able to --

A. I can't -- I would have to have the transcript in front of me and refer to a particular area.

Q. All right. Are --

A. I'm not too sure we've dealt with that.

Q. Are you able to rank the importance of socioeconomic status in terms of its strength as a risk factor compared to maternal smoking for the production of any of the health end points that you've identified in Exhibit 6?

A. I will state that socioeconomic status is a risk factor for those items in Exhibit 6 that I've identified with socioeconomic status. And smoking is another risk factor for all of the items that I identified in Exhibit 6.

Q. All right. But in terms of quantifying the size or the magnitude of that risk factor, socioeconomic status, and comparing it to maternal smoking, you're not prepared to do that?

A. Correct.

Q. Is marital status a risk factor for

spontaneous abortions, reduced birth weight and premature births?

A. Indirectly, in that most people who are married are older than 19 as opposed to unmarrieds, which are frequently teenagers, and teenage age is a risk factor for prematurity, low birth weight, spontaneous abortions.

Q. And is it true, then, that being unmarried is a risk factor for spontaneous abortions, reduced birth weight and premature birth, simply -- through the effect of age or younger age, that unmarried mothers tend to be younger? Is that what you were saying?

A. That's what I just said.

Q. Okay. You mentioned yesterday, and I just want to make sure it embraces all three of these classifications, young maternal age below 19 and older maternal age above 35.

A. I think those are the numbers I used, yes.

Q. All right. Are both of those age classifications risk factors for spontaneous abortion, reduced birth weight and premature birth?

A. I'm not positive regarding the risk of the

older patient for abortions, but I would -- I think it probably very well is, because chromosomal abnormalities increase with age above 35 and those frequently result in abortions. But certainly it is known that older patients have smaller -- may have as a group smaller babies and more prematures.

Q. And with respect to mothers who are 19 or less who deliver babies, that is a risk factor for spontaneous abortions, reduced birth weight and premature births?

A. Asked and answered.

Q. Okay. And when you say that, you mean yes, right?

A. No, I mean you've already asked the question and I've already answered it.

Q. Okay. But I -- is the answer to that question yes?

A. I've already answered that question.

Q. I would appreciate it if you would answer it again. I'm sorry.

A. You've asked once again whether at age less than 19 is associated with prematurity, low birth weight and abruptio -- abruptios?

Q. Yes. No, no, spontaneous abortions, reduced

birth weight and premature births.
A. Age under 19 is associated with those three entities, as I've already stated.
Q. All right. And is it age 19 or below or below 19?
A. Depends on how you calculate 19, probably. Nineteen typically is celebrated at the end of the nineteenth year. So if you say -- I don't know whether the date is defined as after the 19th year or during the 19th year or before the 19th year.
Q. Have you seen the literature that has investigated the relationship between wantedness at conception? Are you familiar with that term, whether or not a baby was wanted when it was conceived?
A. I know there are literature that deal with that issue, and I'm not prepared to give an opinion.
Q. Whether it's a risk factor for any of the health effects?
A. I don't know.
Q. Okay.
A. That's an obstetrical question.
Q. Are -- is non-White race and specifically

Black race a risk factor for each of the health effects that you've listed in Exhibit 6?

A. No.

Q. All right. For which is Black race not a risk factor?

A. Well, I don't think there is any data that would suggest that Blacks as a group have more mental retardation than other races. I don't think that Black as a single entity addresses the carbon monoxide or nicotine issues. I don't think that -- let's see. I'm not sure about the placental injury issue.

MR. MINTON: Let's go off the record.

THE VIDEOGRAPHER: The time is 8:24 a.m. We're going off the record.

(Discussion off the record.)

THE VIDEOGRAPHER: The time is 8:45. We're on the record.

Q. (By Mr. Minton) Dr. Speer, we just had a brief off-the-record discussion. And what I've agreed to do is to try to identify with you what the health effects are that you've listed in Exhibit 6 and -- so that we can put

them up on the board and you'll have a convenient means of looking up and seeing them, if you need to sort of review the list, rather than trying to ask you to recall from memory each time each of the health effects that are listed in Exhibit 6.

So what I would like to do with your help is to go through and get a list of what has been identified in Exhibit 6. Okay?

A. All right. You have Exhibit 6 before you?

Q. I have what I think is a list. And what I would like you to do is go through that with me so that Mr. Blevins can then write down what we agree to. Okay?

A. All right.

Q. All right. In the category adverse pregnancy outcomes for the neonate --

A. Correct.

Q. -- there is listed spontaneous abortions, reduced birth weight, premature births, abruptio placenta and placental injury, correct?

A. Correct.

Q. All right. Then in a category that deals with mechanisms -- is that a fair statement --

the next paragraph that deals with mechanisms, you talk about how the fetal effects of all chemicals found in tobacco are not completely known?

A. Correct.

Q. You talk about carbon monoxide binding preferentially to fetal red blood cells?

A. In preference to oxygen.

Q. In preference to oxygen with accentuated hypoxemia?

A. Correct.

Q. You talk about nicotine acting as a potent vasoconstricting agent decreasing uterine blood flow?

A. Correct.

Q. All right. And there is a mention made of direct fetal effects of nicotine?

A. Because nicotine is found in higher concentrations in the fetus than in the mother.

Q. All right. And under infant complications, you mention studies that show a relative risk of 1.75 for mental retardation in children of mothers who smoke?

A. Well, let's phrase it as it's written: A 75

percent increase in the prevalence of mental retardation in the mothers who smoke.

Q. All right. And then you mention other studies that have shown behavioral problems in children of mothers who smoke raising concern over neuro-developmental disorders?

A. Correct.

Q. And then finally there is reference made to an increased risk of SIDS in children of mothers who smoke, correct?

A. Fourfold increase, correct.

Q. All right. Then finally, you discuss that infants born of mothers who stop smoking during the first trimester have -- and I'm going to shorten this and if I shorten it improperly, say so -- have pregnancy outcomes that are more in line with infants of mothers who didn't -- do not smoke?

A. Correct.

Q. All right. And that mothers who begin smoking during pregnancy have similar fetal effects to those found in infants whose mothers smoked throughout pregnancy?

A. Correct.

Q. All right. And is that exhaustive?

A. That's what's in Exhibit 6.

MR. BLEVINS: In fairness, yesterday the doctor also identified asthma or the asthma and upper respiratory infection stipulation, I guess, or however he phrased it. But just in fairness to you, that was identified yesterday outside the context of the report.

MR. MINTON: All right. And we had a discussion about asthma yesterday as well.

MR. BLEVINS: Yes.

THE WITNESS: Correct.

MR. MINTON: All right.

MR. BLEVINS: I'm not sure you can read that, but at least it was a shot.

THE WITNESS: It's colorful.

Q. (By Mr. Minton) With asthma added up there --

A. Which really isn't part of Exhibit 6.

Q. All right. Is it really part of something that you intend to testify about?

A. If asked.

Q. All right. But is that -- have we then gone through all of the various areas about which you intend to testify?

A. That are in Exhibit 6, correct.
Q. All right. Well, is there anything else?
A. I don't know. People ask me questions all the time.
Q. All right.
A. And it's entirely possible that you or your colleagues or Mr. Blevins and his colleagues will think up new and interesting questions to bring to my attention that have not been exhaustively dealt with yesterday and today.
Q. Okay.
A. So I can't guarantee you guys.
Q. All right. But in terms of what you've prepared yourself to testify for in connection with your opinions in this case, what we've just identified is it, correct?
A. That's not what I said. I said that if you or your colleagues or Mr. Blevins and his colleagues ask me other questions that have not been addressed yesterday and today, I am prepared to give an opinion, if I have knowledge that warrants an opinion.
Q. But in terms of what you've been asked to do, that's it, is it not?
A. Up to this point, yes.

Q. Okay. No one has asked you, for instance, to testify about respiratory distress syndrome, correct?
A. As an effect of prematurity, I've already given an opinion about that.
Q. And as I recall your opinion yesterday, you don't know whether or not respiratory distress syndrome is or is not associated with maternal smoking, correct?
A. I don't think you asked that question. We talked about respiratory distress syndrome as being a consequence of prematurity.
Q. All right. Do you know whether or not studies have investigated if respiratory distress syndrome is associated with maternal smoking?
A. I am unaware of such study -- such studies.
Q. And therefore unaware of any association positive or negative?
A. At the present time, correct.
Q. All right. Now, when we -- before we created the list, you said something that you did not believe that mental retardation was associated with race. Did I hear that correctly?
A. Correct.
Q. All right. And by that, did you mean that you

are unaware of any studies that have identified race as a risk factor for mental retardation?

A. No. I said that mental retardation is not associated with being of a specific race --

Q. All right.

A. -- period.

Q. Is race a risk factor for mental retardation?

A. Based on what I just said, then, no, not as a -- not as race by itself.

Q. All right. So you have not seen any study in which race has been identified as a risk factor for mental retardation?

A. I think you're trying -- you're twisting words. Patients of given races, say American Indians, have a higher incidence of alcoholism and other medical problems that do result in a higher incidence of mental retardation, but it's not because they are American Indians.

So, yes, you can say that in American Indians there is a higher incidence of mental retardation. Studies certainly have said -- made those statement. But studies have not made statements, to my knowledge, that race is an independent factor and causal

for mental retardation, whether you be Black, Green or Purple.

Q. Okay. You just said, as I understand it, alcohol consumption is a risk factor for mental retardation?

A. Correct.

Q. And so in any study, we would expect to see as alcohol consumption increases, the incidence of mental retardation to increase, correct?

A. Correct.

Q. Race and ethnicity are associated with spontaneous abortions, reduced birth weight, premature births and placental injury, are they not?

A. No.

Q. Is there a higher incidence?

A. Not by -- not because of race or ethnicity. There may be other factors that cause some of those problems that you enumerated, but not race or ethnicity.

Q. All right. Is there a difference between the rate of spontaneous abortions when comparing White populations to Black populations?

A. I believe there is.

Q. And it's higher in Black populations, is it

not?
A. Correct.
Q. And when controlled for all known confounders, does that higher association persist?
A. I don't know.
Q. All right. Is there a higher incidence of -- or prevalence of reduced birth weight among Black mothers as opposed to White mothers?
A. We already addressed that yesterday.
Q. And the answer is yes, isn't it?
A. Correct.
Q. All right. And does that difference persist when controlled for all known confounders?
A. That, I believe, is a true statement.
Q. All right. And would it be true to say that you don't know what the magnitude of the risk difference is between Whites and Blacks?
A. I think we addressed that yesterday.
Q. And the answer is no, you don't, correct?
A. Correct.
Q. All right. Is -- is there a difference between the incidence or prevalence of premature births among Black mothers compared to White mothers?
A. I don't know.

Q. Is there a difference in --
A. And you're speaking purely on the basis of race again?
Q. Yes.
A. Okay. Just wanted to make sure we're clear.
Q. Well, are you suggesting that you're aware of data which shows that a crude odds ratio says yes but then when adjusted for confounder says no?
A. I think I may have seen data that suggests that.
Q. Okay. Do you know where that data came from?
A. If I could tell you where all the data I've learned over 25 years came from, I could probably make a lot of money.
Q. Is placental injury, either in the form of abruptio placenta or any other form of placental injury, associated with race?
A. Strictly speaking, abruptio placenta is not placental injury in the definition that I'm using in Exhibit 6.
Q. All right. Then let's break it down into two. Is abruptio placenta associated with race, the incidence or prevalence of abruptio placenta?

A. Again, that's an obstetrical question. My belief and knowledge is that although abruptio placenta occurs more frequently in perhaps the Black population, the White population, when you account for age, it -- that difference may well disappear.

Q. You specifically identified cocaine yesterday as a risk factor for a variety of adverse pregnancy outcomes, right?

A. Particularly abruptio placenta.

Q. Is cocaine use a risk factor for spontaneous abortions?

A. Yes.

Q. Is cocaine use a risk factor for low birth weight babies?

A. Yes.

Q. Is cocaine use a risk factor for premature births?

A. Yes.

Q. All right. Do you have knowledge of what the prevalence of cocaine use during pregnancy is of mothers whose -- who deliver babies and whose care is paid for by Texas Medicaid?

A. No.

Q. Have you-all done surveys at any of the

hospitals with which you are associate -- associated to investigate the prevalence or incidence of cocaine use among pregnant mothers in any population?

A. Ask my obstetric colleagues. You have an expert on your staff that probably could give you that answer.

Q. All right. But you're not aware of any such data?

A. I think there have been some surveys, but I don't know the extent of them and I don't know the results of them.

Q. All right. Are you aware of data that shows that when asked, pregnant mothers underrepresent their cocaine use?

A. They underrepresent every use of anything, including cocaine.

Q. Is cocaine use associated with mental retardation? Is maternal cocaine use associated with mental retardation in a baby?

A. I would anticipate it probably is.

Q. All right. Is alcohol use associated with spontaneous abortions?

A. Don't know.

Q. Reduced birth weight?

A. Yes.
Q. Premature births?
A. I believe so.
Q. Mental retardation?
A. Asked and answered. Yes.
Q. Other neuro-developmental disorders?
A. As a general catch-all, yes.
Q. SIDS?
A. Yes.
Q. What other drugs besides cocaine and obviously in your opinion maternal tobacco smoking are associated with adverse pregnancy outcomes?
A. Alcohol.
Q. All right. Excluding those three, what else?
A. There are a variety of pharmacological agents that are used to treat conditions in the mother that have an association with adverse fetal outcomes.
Q. Are there other illegal drugs or drugs of abuse?
A. Amphetamines have been associated with an untoward fetal outcome.
Q. Is caffeine associated with adverse pregnancy outcome?
A. There are a number of studies debating that

issue. I don't think that's been finalized.
Q. All right. You haven't made up your mind one way or the other?
A. Correct.
Q. Is employment during pregnancy a risk factor for adverse pregnancy outcome?
A. What kind of employment?
Q. Well, let's say that it is the type of employment that is sufficient to create repeated instances of fatigue.
A. Mental or physical --
Q. Physical.
A. -- fatigue?
Q. Physical.
A. If you're talking about being on your feet all day or at a manual labor type of employment, yes, that has been shown to be a risk factor for prematurity.
Q. How about with abruptio placenta or placental injury?
A. I don't think placental injury. Perhaps abruptio. I don't know.
Q. How about reduced birth weight?
A. Don't know.
Q. Is education level of the mother something

that's been identified as a risk factor for adverse pregnancy outcome?

A. There is an association between education and socioeconomic status and adverse pregnancy outcomes. But again, you must correct for those persons who have lower educational attainment who can't go further and those persons who could. And those persons who could, education by itself probably has no bearing.

Q. And when you say "could go further," you mean that they achieved a level of education consistent with their abilities and were unable to go further in the educational system?

A. Because of outside influences they have no control over, or they might have control over but because of their peer group and other factors, did not go further. But they are not intellectually impaired. That group has no particular reason to have children that are intellectually impaired.

Q. All right. Well, is --

A. What I'm saying is, education by and to itself, you know, once corrected, probably has

little bearing on the issue.

Q. All right. Is education -- has education been associated with prematurity or low birth weight?

A. Education un and to -- and by itself, acts like race.

Q. Could you expand on that?

A. If you have only education as the variant and you've excluded other issues such as drug abuse, infections, other issues -- other items that have a stronger association with adverse outcome with -- and you've corrected for mental abilities, then education in and by itself probably has very little bearing on anything.

Q. Is low maternal weight gain a risk factor for low birth weight?

A. It would depend on what caused the decrease in maternal weight gain.

Q. In other words, you've seen data which have stratified causes of low maternal weight gain and found some of those associated with low birth weight and some not?

A. For example, low maternal weight gain secondary is associated with the smoking of

cigarettes and those babies are smaller and born more prematurely and have the problems that we've just talked about.

There are some populations, however, and some individuals who have no risk factors except that they don't gain a lot of weight. And if you took that population, I'm not too sure that it has or has not shown adverse outcome.

Q. In other words, after controlling for cigarette smoking, you're not sure whether or not --

A. Or other factors that influence maternal weight gain.

Q. You're not sure whether the association persists after you control for those factors?

A. Correct.

Q. Is maternal nutrition a risk factor for adverse pregnancy outcome?

A. Only if the mother is starving.

Q. And just so we've got an understanding of what you mean by starving or starvation, what level of nutrition?

A. The best studies came out of Amsterdam during World War II starvation situations, and I

don't know -- I can't remember the exact calorie intake, but I think it was less than a thousand calories per day is associated with the lack of fertility and a somewhat diminished birth weight when the population studied. I'm not too sure they looked at other factors such as mental retardation and others, but that data may also be out there.

Q. Is lack of access to or inferior quality of prenatal care a risk factor for adverse pregnancy outcomes?

A. Some people would like to say so, but there is increasing data that would suggest that that may not be as large a factor as we would wish.

Q. Are you saying that it exists as a risk factor but the magnitude of the association has been called into question?

A. Correct.

Q. Is short interpregnancy interval a risk factor for adverse pregnancy outcome?

A. There are some studies that I'm aware of, there may be others that I'm not aware of, that suggests that in some people a short interpregnancy interval is associated with risk of prematurity.

Q. Would it be correct that as to all of the risk factors that we've just identified, you could not rate them one against the other or against maternal smoking in terms of determining how important they are on a population basis?
A. If you gave me a list of everything we've talked about, I would certainly make an effort to rank, but it would be difficult to in some instance to -- instances to rank them as opposed to other instances.
Q. All right.
A. If, for example, you asked me to rank cocaine against the others for abruptio placenta, it would be right up there at the top. That's easy. But in other instances, it would be more difficult.
Q. And how would you go about doing it?
A. If you want to read them off and give me a piece of paper, I'll make an effort to rank them.

MR. BLEVINS: I don't know that I've covered all of them or not.

Q. (By Mr. Minton) Well, let me ask you this, Dr. Speer, before we go through the exercise. Would it be correct to say that you don't know

what the point estimates or the confidence intervals for the relative risks for any of those risk factors might be?

A. Sitting here today not having been given the opportunity to investigate that, nor having the opportunity to investigate that, you're correct.

Q. All right. So any -- any ranking that you give us would be a guess?

A. An informed guess.

Q. But it would not be based on data that would allow you to rank relative risks, correct?

A. It would be data that would allow me to rank.

Q. All right. You would need data that would allow you to rank relative risks before you could go through the exercise?

A. No.

Q. All right. You would need data that would allow you to see what the relative risks were in order for your ranking to be scientifically informed, correct?

A. No.

Q. What would be the scientific information that you would rely on in order to rank them without the knowledge of either the point

estimates or the confidence intervals for the relative risks that have been described in the literature?

A. My knowledge and training over the last 25 years.

Q. All right. And what does your knowledge and training over the last 25 years tell you about the relative risk of socioeconomic status for the production of adverse pregnancy outcome?

A. Socioeconomic status in and by itself may well have little bearing on pregnancy risk and outcome of pregnancy, as I've already stated.

Q. All right. And what does "little bearing" translate to in terms of an estimate of a relative risk point estimate or confidence interval?

A. Okay. You're talking about apples and oranges. I'm telling you I can rank, if you wish me to, the relative risk of a given condition with a given outcome. You're asking me confidence intervals means and relative risks. That I cannot do for you because I do not have the data in front of me.

Q. Well, do you have -- you, I think, just suggested that you know what the relative

risk -- what is a relative risk?

A. A relative risk is a number, not a concept.

Q. All right. And you do not have any of those numbers for any of the risk factors that we've just identified, correct?

A. Correct.

Q. All right. And those numbers would be the means by which you would rate them if you were going to rate them scientifically, correct?

A. If I was going to rate them based on relative risk, that's how I would rank them. I can rank them based on concept. Alcohol is a bad thing. Cocaine is a bad thing. Alcohol is probably the leading cause of mental retardation in this country, according to some authorities.

So if you ask me of "X" list, is alcohol high on the list for causing mental retardation, the answer is obviously yes. I don't need a relative risk to tell you that.

Now, if you want to attach a relative risk, then that is the relative risk of what alcohol's effect is on the issue of mental retardation. Is it a cause of mental retardation? Absolutely.

Q. Dr. Speer, a relative risk is a rate in the exposed -- or a ratio of rates between the exposed and unexposed, correct?
A. The risk of a certain happening occurring due to, for example, alcohol, yes. It's a number.
Q. Well, and do you know where that number comes from, what the construct is that leads to that number? If I told you that the relative --
A. Population studies --
Q. All right.
A. -- of patients who ingest alcohol and the relative incidence of mental retardation, the population study of other variables being dealt with.
Q. And so they compare the incidence rates or ratios between exposed and unexposed populations, correct?
A. Correct.
Q. And determine a risk difference?
A. Correct.
Q. And that would be the means by which one would rate the relative importance of different risk factors, would it not?
A. That's the relative risk, yes.
Q. All right. Is there any other scientific

means in order to compare the relative importance of risk factors?

A. There are other statistical methodologies that are used in similar populations, but your concept is correct.

Q. All right. And none of which you are prepared to go through today for us, correct?

A. I am not prepared to go through numbers, because I don't have the numbers in front of me.

Q. Okay.

A. But I know that the relative risk is higher for mental retardation in populations who consume alcohol when they are pregnant than populations who don't consume alcohol when they are pregnant. That is a statement of fact.

Q. That is a greater than one. The relative risk is greater than one, correct?

A. You're going to confuse the jury by your relative risk of greater than one, but, yes, you're correct.

Q. What is a relative risk of one?

A. There is no risk in populations; they are equal.

Q. All right. And a relative risk of 1.1 means what?
A. Twenty percent risk in the population is exposed, higher.
Q. One point one means a twenty percent --
A. No, you said -- I thought you said 1.2. I'm sorry. If you said 1.1, it's ten percent.
Q. And a relative risk of 2.0 means what?
A. Two hundred -- a hundred percent more.
Q. What do you believe are the constituents of socioeconomic status which best describe it in terms of the effects that socioeconomic status has on adverse pregnancy outcome?
A. Not being a sociologist, I cannot answer your question.
Q. Do low income groups usually include younger and less educated women than do middle income groups?
A. Frequently.
Q. Are Black patients more likely to be poorer, have less education and seek prenatal care later during pregnancies than White -- than White patients?
A. In the population of the United States as a whole?

Q. Yes.
A. Yes.
Q. And are you familiar with any sub-populations in which that is different?
A. I would anticipate in River Oaks there is probably no difference.
Q. So in order to accurately characterize those differences, we need to focus on a particular population and its characteristics?
A. Not necessarily particular population. We need to focus on the population as a whole, not a particular population.
Q. I thought you just pointed out that River Oaks would have different characteristics than the population as a whole?
A. Right. That's why I said the population of a whole is a better sample than restricted or particular populations.
Q. What is the mean income in the United States?
A. I don't know.
Q. Do you expect that it would be substantially different than the mean income in the Texas Medicaid population?
A. Don't know. Not knowing the mean income of the United States and not knowing the mean

income of the Texas Medicaid population, I have no basis to make an opinion.

Q. All right. What's the racial balance of the population of the United States in terms of Blacks and Whites?

A. For the entire country, I don't know.

Q. All right. What is the racial balance of the Texas Medicaid population?

A. Large numbers of Hispanics and Blacks and with a less number of Whites --

Q. Is it similar --

A. -- is my understanding.

Q. -- similar to the racial population of the United States as a whole?

A. I doubt it, because Texas has more Spanish than some parts of the United States as a whole, the proportion.

Q. What's the incidence of cocaine use among the population of the United States as a whole?

A. Don't know.

Q. Do you know how that compares to the incidence of cocaine use among constituents in the Texas Medicaid population?

A. Not having studied cocaine, I don't know.

Q. You have no suspicion whether it would be

higher or lower?

A. Don't know.

Q. Is marijuana use associated with adverse pregnancy outcome?

A. Don't know.

Q. Do you know what the incidence of births to unmarried mothers is among the United States population as a whole?

A. I haven't looked that up recently.

Q. All right. Do you know how it may or may not compare with the incidence of births to unmarried mothers in the Texas Medicaid population?

A. I have no data to give you an answer one way or the other.

Q. Do you know what the incidence of substance or alcohol abuse is among mothers in the general -- pregnant mothers in the general United States population?

A. No.

Q. Do you know how that may compare to the incidence of substance or alcohol abuse in the -- among mothers in the Texas Medicaid population?

A. No.

Q. Do you know what the concept of external validity is in epidemiology?
A. I've heard the term. I don't know exactly what its definition is.
Q. Do you know the criteria by which epidemiologists judge the generalizability of findings from one population to another?
A. As I've already stated, I'm not an epidemiologist nor am I a statistician and I do not know the inner workings of either group.
Q. And I'm going to ask this rapidly because I think it does cover some ground that we've already covered, but I want to make sure it's -- it has been done.

Dr. Speers, as I understand it, you've made no analysis of the Florida Medicaid population or any attempt to compare it to any other population with respect to any factor, correct?
A. First, I'm a singular person; no "S" on the end of my name.
Q. I'm sorry.
A. And you're correct in your assumption.
Q. And that would include income, age, housing

conditions, race, ethnicity, education level, drug use, access to medical care, use of medical care, quality of care, diet, nutrition, type of work performed outside the home, any obstetric history or obstetric risk factors?

A. I have done no investigation that would address any of those issues.

Q. And you've not analyzed the incidence or prevalence of smoking among pregnant mothers in the Texas Medicaid population?

A. No. That's not my job nor my field of endeavor.

Q. Nor have you analyzed the incidence of adverse pregnancy outcomes in the Texas Medicaid population?

A. Correct.

Q. Nor have you analyzed the contribution maternal smoking may or may not have had to adverse pregnancy outcomes in the Texas Medicaid population?

A. Correct.

Q. Nor have you analyzed the contribution of other risk factors to any adverse pregnancy outcomes in the Texas Medicaid population?

A. Correct. As I stated earlier, I have done no analyses on any population.
Q. Dr. Speer, you mentioned in your -- in Exhibit 6 mental retardation. What study have you done of mental retardation during your career?
A. I have not done a study on mental retardation in my career.
Q. All right. You haven't written on it or lectured on it?
A. I've lectured on it insofar as it exists. There are certain findings that are associated with adverse intellectual outcome, if that's what you mean.
Q. Lectures given to whom?
A. Medical students, residents, conferences.
Q. Describing what?
A. Well, for example, a ventricular hemorrhage has an associated neurologic disability of X, Y and Z. We know that certain categories of prematures have a much higher incidence of mental retardation than other categories of prematures.
Q. All right. In certain portions of Exhibit 6, you use the verb "caused." In certain

portions of Exhibit 6 you do not use the verb "caused." And let's get an understanding. Is it your opinion that maternal smoking causes mental retardation?

A. A cause. It may be -- it looks like it may be a very important cause, but it is a cause.

Q. All right. And is that based on one study that you have reviewed?

A. There are a couple of studies.

Q. You've given us a copy of the Drews study which was --

A. That's one of the most recent, correct.

Q. All right. What other studies are there?

A. I can't name them, but they are there.

Q. All right. Describe what you know about the other studies that are there.

A. The conclusion is that smoking is associated with mental retardation.

Q. All right. What group was studied -- is it more than one other study or one other study?

A. It's my understanding there are more than one other studies.

Q. Have you read them?

A. I have seen abstracts of them. I probably have read a few of them over the years, yes.

Q. But as you sit here today, you can't give us any more information about them than that?
A. Correct. I would refer you probably to the reference list in the Drews article. That would probably be a good starting place.
Q. All right. Did Drews -- did Drews mention three studies which had failed to find an association between maternal smoking and mental retardation?
A. May I have Drews article?
Q. I believe it's behind you marked as an exhibit.
A. And perhaps you would draw my attention to the correct paragraph.
Q. I believe it's over on the first page in the second column near the bottom where she mentions three studies which have specifically looked for an association between maternal smoking and mental retardation.
A. Okay. There are three studies noted.
Q. All right. And does she -- does she say that in each instance there, that the authors were unable to find a statistically significant difference of mental retardation in the children of mothers who smoked?

A. Given the definitions that are expressed, yes.
Q. All right. And so those three that she refers to there are negative studies, correct?
A. In that they did not find an association with what they defined as mental retardation, correct.
Q. All right. And does she mention any study in which it is alleged to have been found an association -- a statistically significant association between maternal smoking and mental retardation?
A. She mentions 13, 14, 15, 16, 17, I think, studies that are consistent which examine the effect of smoking on cognitive function.
Q. Right. But those didn't examine the relationship between smoking and mental retardation, did they?
A. What is your definition of "mental retardation"?
Q. Well, let's start with yours. How do you define "mental retardation"?
A. Mental retardation is a diminished cognitive function, to use this terminology.
Q. All right. And how is diminished cognitive function -- how is cognitive function

characterized, Dr. Speer?

A. Measures whatever you're measuring to define cognitive function. Frequently it may be Ballian examinations or other examinations in the young child. It may be SAT score. It may be a more formal, I think it's Minnesota Intellectual Test. I can't remember the exact name. There are several standardized tests that are used in this country to measure ability to think and reason.

Q. All right. The Minnesota Multiphasic Personality Inventory, is that what you're referring to?

A. That may be. It's not -- this is not my field, so I don't know all of the various tests that are currently available.

Q. What is -- psychometric testing is not your field?

A. I'm a neonatologist, sir. I think we made that clear earlier.

Q. Do you know -- are you -- do you consider yourself an expert in psychometric testing?

A. No.

Q. All right. Do you know what the product of psycho -- how the product of psychometric

testing is characterized?

A. I'm sorry?

Q. I'll start over.

A. Please.

Q. What is IQ?

A. Intellectual quotient.

Q. And how is that defined?

A. It's defined by a variety of standardized tests that measure what is termed intellectual quotient.

Q. Well, that's -- that's kind of a circular response. What is an intellectual quotient? What is the -- a quotient implies that one number is being divided by another, correct?

A. Yes.

Q. Do you know what is being divided by what?

A. I am -- I don't do these types of -- I don't study intellectual quotients.

Q. All right. Do you know what number is being divided by what other number?

A. No.

Q. All right. Do you know what "G" is in psychometric testing?

A. No.

Q. Do you know how well psychometric tests

measure true cognitive function?

A. How they measure true cognitive function?

Q. How well they are estimated to measure true cognitive function.

A. I don't do these tests. I don't have any knowledge on their development. I have no knowledge on what validation criteria they have met to be used. I would refer you to someone who does.

Q. Have you read any of the negative studies that Drews refers to there?

A. Well, her use of negative is on Page 1, as you correctly pointed out. She then reviews the three negative studies and has some criticism of them. I have not personally reviewed any of the three.

Q. All right. Have you reviewed Drews in terms of the study methodology that was used?

A. In a -- I read this paper a number of months ago when it first came out, which I believe is -- what is the date? '96. I can't remember when it was published. I apologize.

I read the paper when it first was published. I have not reviewed it in-depth since that point in time, but my

impression at that point in time was that it was a quite exhaustive study and was population based and had adequate numbers and indeed would appear to be a fairly strong statement, as a single statement.

Q. All right. Dr. Speer, what percentage of IQ is considered to be heritable?

A. I don't know the exact percentage these days. Certainly inheritance and genetics plays a role in what you are given to start out with.

Q. Do you know what the -- do you know if there have been studies that have investigated the heritability of IQ, studies, for instance, of monozygotic twins raised separately?

A. I believe there are such studies.

Q. All right. And they demonstrate, do they not, that there is a substantial component of IQ that's heritable, correct?

A. That's what I've said, yes.

Q. All right. And -- but in terms of telling us approximately what percentage of IQ is heritable, you're not prepared to say at this point?

A. Correct.

Q. All right. A baby inherits its IQ from -- a

part of its IQ from its mother and its father, correct?

A. That is the conventional wisdom, correct.

Q. All right. That was not controlled for in Carolyn Drews' study, was it, Doctor?

A. What the IQ of the parents were, is that what your question is?

Q. Was IQ of either the mother or the father controlled for in that study in terms of its determinate of the IQ of the baby?

A. I don't believe they addressed that issue, but the study is a population-based large study and it probably is irrelevant.

Q. What is irrelevant?

A. Whether the IQ of the parents was in this study.

Q. Well, we've established that IQ of the parent is a strong determinate of the IQ of the baby, correct?

A. As a statement, yes.

Q. All right. And Carolyn Drews, in her study, did not control for maternal or paternal IQ, did she?

A. She doesn't have to.

Q. Why doesn't she have to?

A. Because she has a population-based study. A population-based study, if it takes across all the population, that variable disappears. That's the beauty of a large population-based study. That's why you don't do studies with a single hospital or a single home for mental retardation or what have you. You do a population-based study.

Q. Okay. Let's say I designed a study like this. All right. I took -- I took children that I knew had mental retardation. All right?

A. Okay.

Q. I'm going to need you to focus on this. All right?

A. Okay. I am.

Q. And then I compared them to children who did not have mental retardation, correct?

A. All right.

Q. You're with me so far?

A. I'm with you.

Q. All right. On that basis, based on what I have done, I should assume that there's a different parental level of IQ between the parents -- between the parents of the retarded

children and the parents of the non-retarded children, correct?

A. You're stating that --

Q. No, I'm designing a new study here.

A. Okay.

Q. All right. And what I've done is, I've taken children who I know are retarded. All right? And I've taken as my cases. And in my comparison group, I have taken children who are -- I know are not retarded. And I'm talking about idiopathic mental retardation -- all right -- for which there is no explicable condition. All right?

A. Uh-huh.

Q. On that basis alone, statistically I know that the IQ of the parents of the mentally retarded children on a population basis will be different from the IQ of the parents of the non-retarded children, correct?

A. In the way you've designed your hypothetical study, yes.

Q. All right.

A. But that's not how they designed this study.

Q. Is the quality of the home environment a determinate of IQ in a child?

A. It certainly may be a determinate.
Q. Is parenting style a determinate of the IQ of a child?
A. How do you define "parenting style"?
Q. Well, let's start with an abusive or neglectful home.
A. That may have an influence on the ultimate intellectual and scholastic attainment of the child, correct.

MR. MINTON: I'm sorry, did he say intellectual and scholastic attainment?

THE COURT REPORTER: Yes.

THE WITNESS: You're not supposed to nod. You're supposed to say "yes" or "no."

THE COURT REPORTER: Thank you.

Q. (By Mr. Minton) Are smokers better or worse maternal care-givers than nonsmokers?
A. Don't know.
Q. Has anyone, to your knowledge -- did Drews suggest a hypothesis regarding any possible mechanism for the mediation of a cognitive effect for maternal cigarette smoking?
A. In a quick re-review of her article, I don't believe that she proposes any data or

hypothesis on how.

Q. All right. You have forcefully told us that you think alcohol use is the single most important determinate of mental retardation in children insofar as maternal exposure, correct?

A. As I said, several authorities feel that that is the single -- the most important cause for mental retardation. It's not what I said.

Q. And do you think it is?

A. I think it is certainly a major cause of mental retardation. I don't know if it's the number one cause of mental retardation.

Q. And as I understand it, you said quite forcefully that you did not think that race had been associated with mental retardation?

A. Race by itself, and only by itself. I don't believe that Black people are less intelligent than any other race, nor do I feel that White persons are less or more intelligent than any other race, et cetera.

Q. Are Blacks at higher risk compared to Whites for mental retardation?

A. If you're saying -- if you're asking that in a population of Blacks is mental retardation

higher than a population of Whites of the same -- all other factors being controlled, including the incidence of low birth weight, small-for-dates babies, use of alcohol, tobacco and other products, I'm not too sure that that statement is a true one. But if you don't control for them, yes, it is a true statement.

Q. Okay. So in some crude odds ratio, in other words some unadjusted odds ratio, you may expect to see Black race emerging as a risk factor for mental retardation, but once you adjust or control for a variety of variables that you just named, then that increased risk disappears?

A. It may disappear. And again, I have not studied this issue, I have not analyzed this issue. I do not do research in this area. I'm giving you an opinion based on what reading I have done.

MR. BLEVINS: Doctor, what time do you need to leave here in order to make your 10:00 meeting?

THE WITNESS: About another 15 minutes. It's close.

MR. MINTON: In about five I'm going to be at a convenient point for a break. But I don't -- you know, we can do whatever.

Q. (By Mr. Minton) What are the risk factors for mental retardation in children?

A. Goodness gracious. There may well be literally hundreds of them. I don't purport to be an expert on all, nor can I probably name all of them.

Q. Is anemia?

A. Anemia, per se? Anemia, per se, may not be. Iron deficiency anemia may be.

Q. Is toxemia?

A. Toxemia of whom?

Q. The mother.

A. Eclampsia?

Q. We'll get to eclampsia. How about toxemia without eclampsia?

A. Probably as a global statement.

Q. All right. By the way, is -- is maternal smoking negatively associated with toxemia?

A. I do not know.

Q. Are preeclampsia and eclampsia associated with mental retardation?

A. Preeclampsia is toxemia. We've already answered that. Eclampsia is a seizure during the process worsening the preeclampsia. Seizures in mothers are not good for babies. So the answer would be yes.

Q. Is low weight gain in pregnancy a risk factor for mental retardation?

A. I think we've addressed that issue before.

Q. Well, my recollection was that it was with respect to other health end points.

A. You're asking specifically is poor weight gain associated with mental retardation?

Q. Yes. Poor maternal weight gain during pregnancy.

A. Depends on the cause of the poor maternal weight gain.

Q. So it might be or it might not be?

A. Correct.

Q. Is a maternal urinary tract infection in pregnancy associated with mental retardation?

A. Don't think so.

Q. How about if it's a complication which is linked to endotoxemia and fetal leukoencephalopathy?

A. Well, then you're talking about more than just

a simple urinary tract infection, aren't you?

Q. If that's the case, is it then linked with mental retardation?

A. If you're talking about a systemic infection on the part of the mother that originated in the urinary tract that results in the production of endotoxins that can freely pass in the fetal compartment, the answer is yes.

Q. All right. Is chorioamnionitis associated with mental retardation?

A. There are some beginning data that suggests that that's the case.

Q. Is male sex associated with mental retardation?

A. I think in general boys are not quite as smart as young ladies or older ladies.

Q. Does that mean yes?

A. I don't know whether it's mental retardation.

Q. Well, by the definition Drews uses, any -- any risk factor that results in a shift of the intelligence curve will be a risk factor for mental retardation, won't it?

A. I don't remember reading that. It is entirely possible boys have a higher incidence of mental retardation because they have a higher

incidence of respiratory distress syndrome, there are more of them that are premature, and so certainly those survivors, because they are premature, and prematures have a higher incidence of mental retardation than term babies, and there are more baby boys that are premature than baby girls, then baby -- then boys will have a higher incidence of mental retardation. Whether that's based on the fact that they are boys or not, I can't tell you.

Q. What's the mean birth weight differential that's been ascribed to maternal cigarette smoking?

A. You asked that and I couldn't tell you. Remember?

Q. Do you know if it has been said to be of clinical significance?

A. It appears to be of clinical significance because a lot of people say it is, including the Academy of Pediatrics.

Q. Is socioeconomic status a risk factor for mental retardation?

A. It depends on what is the cause of the low socioeconomic status.

Q. Well, if the -- if the socioeconomic status

that is measured is simply census tracked income, is socioeconomic status associated with mental retardation?

A. I stand by my previous answer.

Q. Well, I'm just interested if you know whether or not studies say if we look at populations and if we look at -- and compare them by census track as a measure of socioeconomic status, is socioeconomic status a risk factor or predictor of mental retardation?

A. If you're only going to use that one single rather crude indicator of mental retardation, yes, there is an association between socioeconomic status based on census trackers income and mental retardation.

Q. All right. When we broke yesterday, we were having a discussion about trying to predict back and forward. And I think that you told us twice yesterday that if we have an outcome as to which there were multiple risk factors present, that there is no way, looking backward from the event, to say which risk factors may have contributed in all or part to the outcome, correct?

A. That wasn't the end of yesterday's

discussion. It was about halfway through yesterday's discussion and again at the beginning of today's discussion.

Q. All right.

A. But that's of no real mote.

Q. All right. But with those qualifiers -- I got that part right. We're going to go on to the next part.

A. I think you did.

Q. All right. The -- and what I understood you to say, that going forward, it is your opinion that the use of risk factors can tell you something about predicting risk in an individual. Did I get that right?

A. I think you did.

Q. All right. And that would be done formalistically or in the medical -- in the medical profession through the use of clinical risk prediction scales, correct?

A. I don't know. Remember we talked about -- I know you asked me about clinical risk prediction scales used by obstetricians and I told you that I was not familiar with those and referred you to my obstetrical colleagues for clarification.

Q. All right. Do you know, for instance, whether there is any clinical risk prediction scale for any of these adverse health outcomes that says on the basis of maternal smoking alone, that predicts an adverse pregnancy outcome on the basis of maternal smoking alone?

A. Given that I just told you that I don't know or don't -- and don't use any of those clinical risk prediction scales that you have mentioned, it would be absolutely impossible for me to give you an answer to your follow-up question.

Q. Would it also be impossible for you to say what the predictive power of the combined risk factors that we are aware of are? In other words, if we use the existing knowledge about what risk factors we have and try and predict an event that's going to occur in the future, how often we may correctly predict things?

A. I'm not even sure I know what you're talking about, sir.

Q. Well, a clinical risk prediction scale is a means by which we can take certain risk factors --

A. Excuse me, if I may interrupt. I don't know

about clinical risk prediction scales. Any discussion that you start or try to forward on clinical risk prediction scales is counterproductive, because I can't answer the questions that you're posing.

Q. All right. So you would not know how clinically we would take potential risks to an unborn fetus and attempt to predict or project those risks based on any existing criteria that we have?

A. That's not what I said. I said I cannot use -- I don't know anything about clinical risk predictive scales. I do not know whether those predictive scales do or do not take into consideration smoking.

However, I can tell you that if you take a population and you have a risk of 20 percent higher in a population who smokes as opposed to a population who doesn't smoke, or in the case of low birth weight that appears it's 25 percent higher, then there is a 25 percent higher risk in the populations who smoke of low birth weight compared to the population who doesn't. I can tell you that.

Q. All right. So it's your opinion that there is

an increased risk of 25 percent among mothers who smoke, correct?

A. Well, I'm taking data that is in Exhibit No. 12 that says that if you stop smoking, you decrease the incidence of low birth weight, which is low birth weight, as much -- by as much as 25 percent. If you reduce it by 25 percent, if that figure is correct, then there is a 25 percent increased risk of low birth weight in people who do smoke.

Q. All right. And that is the sole data that you have in order to ballpark a risk of low birth weight among mothers who smoke?

A. If you're talking about data as you have been talking about it as opposed to how I've been talking about it, correct.

Q. Well, I mean, yeah, data in terms of having a hard number to provide us.

A. Correct.

MR. BLEVINS: Good time to break?

MR. MINTON: That's fine.

MR. BLEVINS: Okay.

THE VIDEOGRAPHER: The time is 9:58 a.m. We're going off the record.

(Brief recess.)

THE VIDEOGRAPHER: The time is approximately 11:11 a.m. We're on the record.

Q. (By Mr. Minton) Hi again, Dr. Speer.

A. Good morning.

Q. Dr. Speer, would it be correct to say that no one knows by what mechanism smoking may mediate the effect of prematurity?

A. I'm not too sure that no one knows, but I don't know.

Q. All right. Have you seen any literature in which an author has claimed that they had identified to some degree of scientific certainty that the mechanism of smoking mediated prematurity was "X," whatever "X" may be?

A. I'm unaware of any data.

Q. As I gathered from your testimony yesterday, it is your impression from leading -- reading the literature that the mechanism that you have seen identified for maternal smoking mediated low birth weight is nicotine?

A. That is one possible explanation, yes.

Q. All right. And is that a possibility that is

somewhere below the level of certainty in terms of your opinion regarding what the mechanism of maternal smoking mediated low birth weight may be?

A. Given the number of chemicals that have here to date been identified within tobacco, it wouldn't surprise me that it's a multifactorial issue as opposed to a single agent.

Q. All right. Would it be fair to say, then, that you're not giving an opinion to a reasonable degree of scientific certainty that nicotine is the mechanism by which maternal smoking produces low birth weight?

A. I'm giving the opinion that nicotine may well not be the only cause. It may be a contributing cause, but I don't believe it's probably the only cause.

Q. All right. Are you saying to a reasonable degree of medical certainty that it is a constituent cause of the mechanism by which maternal smoking mediates a low birth weight effect?

A. The information to date would suggest that it is a cause.

Q. That it is a mechanism?
A. It is a part of the total cause.
Q. What are the risk factors that you've seen identified for Sudden Infant Death Syndrome?
A. I think I've noted already that race does appear to have an independent effect on Sudden Infant Death Syndrome. Tobacco appears to increase Sudden Infant Death Syndrome risk fourfold over baseline, no matter what race is involved. Position of the infant appears to play a role in the incidence of Sudden Infant Death Syndrome.
Q. Sleeping position?
A. Sleeping position. Soft bed materials have been implicated in some studies and not in others as a risk in Sudden Infant Death Syndrome. CO2 responsiveness appears to be a risk factor in certain infants with immature respiratory centers. And there are undoubtedly others.
Q. What does CO2 responsiveness mean?
A. That means the ability to initiate breath on the -- with increasing CO2 in the bloodstream.
Q. Has body temperature been associated with SIDS?

A. I'm not too sure body temperature has. There has been at least one or two studies that I'm aware of that relates to environmental temperature.

Q. And do they show an increased risk if the environmental temperature is kept low?

A. No, increased risk if environmental temperature is kept high, if I remember correctly.

Q. Is -- have there been studies which have looked at breast and bottle feeding in terms of the risk of SIDS?

A. Probably. I'm not directly familiar with those studies.

Q. All right. Now, you've given us in your opinion document and also orally here today a relative risk of four. Do you know what study that came from?

A. No, I do not.

Q. Do you know if there is more than one study that reports a relative risk of four?

A. I don't know if there are more than one study or not. I think that there are, but I can't assure you absolutely.

Q. Do you -- do you know -- you don't know where

that four came from?
A. Not off the top of my head, no.
Q. It was just a number that occurred to you as you were writing that document?
A. As I wrote the document.
Q. You didn't consult some other document, then?
A. Not at that time, no.
Q. All right. Nor since?
A. No.
Q. All right. Is there any known causal mechanism for SIDS?
A. Well, one cause, as I implied, may be the responsiveness of the respiratory center to increasing CO2. That appears to be a thread that weaves throughout many of the proposed or the -- not proposed but the observed increased incidence. That -- that certainly is one of the proposed mechanisms in regard to cigarette smoke. It appears to be a proposed mechanism for the position findings and the soft bedding findings.
Q. And when you -- is your opinion referring to a risk among children whose mothers smoked while they were pregnant with those children?
A. Correct.

Q. All right. And it doesn't have anything to do with environmental tobacco smoke exposure?
A. It may be additional -- environmental smoke may play an additional role.
Q. Have you studied the literature on environmental tobacco exposure?
A. I have not studied the literature.
Q. All right. And so do you have any opinion on the relationship, if any, between ETS and SIDS?
A. Who?
Q. Do you have an opinion on the relationship, if any, between environmental tobacco smoke exposure and SIDS?
A. I have -- I think that it's been shown that environmental tobacco smoke may well have a relationship to SIDS. I'm not certain as to the exact weight of that environmental tobacco smoke.
Q. All right. Do you know whether there was quantitative exposure data in the study that you're relying on for the 4.0 relative risk regarding maternal cigarette smoke?
A. I do not remember.
Q. Is there any consistent pathology found in

infants who have died of SIDS?
A. More a lack of pathology than a positive pathology.
Q. Other physicians -- and I -- this is perhaps too much slang for you to tolerate at this point, but other physicians have referred to SIDS as a trash can diagnosis in the sense that it is a diagnosis of exclusion where there is -- there are no other explanatory findings. Do you agree with that?
A. I prefer the term "diagnosis of exclusion."
Q. All right. And a diagnosis of exclusion means we don't have any readily apparent cause to blame a particular child's death on; is that correct?
A. That we presently can identify.
Q. All right. And that's how a SIDS diagnosis is made?
A. It's a -- it is a diagnosis of exclusion at this point in time.
Q. All right. Would it be fair to say that there is an awful lot that's unknown about the occurrence of SIDS?
A. I think you could say that.
Q. All right. And would you be able to ballpark

in terms of the cause of SIDS, you know, the percentage of what we know versus the percentage of what we don't know?

A. Not knowing the totality of what we don't know, no.

Q. Okay. Have there been negative studies reported with respect to maternal smoking status and SIDS in offspring?

A. I would anticipate there are.

Q. All right. Do you have any idea of studies other than the one study that you've cited, what the relative risk has been in those other studies where a statistically significant association has been found?

A. As I've stated, I have not done any literature searches in this area.

Q. Doctor, what behavioral problems are you referring to in your disclosure statements that have raised concern over neuro-developmental disorders?

A. It's my understanding that they fall into school behavioral problems, attention deficit and societal behavior problems. And the association in this area is certainly loose.

Q. Does "loose" mean scientifically unproven, as

far as you're concerned?
A. As I noted in my document, studies have shown behavioral problems in children of mothers who smoke raising concern. And it's raised concern. And that's where the level sits at this point in time.
Q. It's below a scientific demonstration of cause and effect, in your opinion?
A. At this point in time, it is probably less than more likely than not.
Q. Would it be fair to say that you've not made any methodologic analysis of those studies?
A. Correct.
Q. And does the same hold true for the SIDS studies as well?
A. I already stated that.
Q. All right. Is tocolytic therapy used in any of the hospitals that you're on staff of to prevent preterm delivery?
A. Yes.
Q. And has there been substantial success using tocolytic therapy in terms of preventing preterm delivery?
A. What do you mean by "substantial success"?
Q. It -- you raise a good point. What has been

the improvement in the delay of delivery or enhancement of gestational age in mothers in whom tocolytic therapy has been provided?

A. It really depends on the practitioner and aggressiveness of tocolytic therapy and the status of the mother's labor when she presents herself for possible tocolytic therapy.

Q. All right. And I take it from -- that from that response, that the -- the intervention can result in zero improvement to substantial improvement?

A. Correct.

Q. All right. And is there some mean or medium level of improvement that you're comfortable in describing?

A. No. I would refer you to an obstetrician to be able to discuss obstetrical issues.

Q. All right. And I'm going to lump the next four categories together, because I have a feeling we may get the same type of response, but are frequent provider contact, continuity of provider, physical examination and patient education by providers important criteria in terms of preventing premature birth?

A. That's a rather broad statement. Would you

mind enumerating the four again?

Q. Frequent provider contact, continuity of provider, physical examinations and patient education by providers.

A. Certainly the last is very important.

Q. Patient education?

A. Correct.

Q. All right. How about physical examinations?

A. Depends on what the physical examination would entail, obviously. And so depending on the physical exam, it might be anywhere from very important to relatively important.

Q. Is continuity of provider important in terms of preventing premature delivery?

A. People -- I'm quoting obstetrical literature. Once again I refer you to an obstetrician for a better opinion. But if my knowledge of obstetrical literature is current, it is less so than people had hoped. It is important, but less so than people had hoped.

Q. Do any of the hospitals with which you're associated provide intrauterine monitoring as a means of forestalling premature delivery?

A. Exactly what do you mean by "intrauterine monitoring"?

Q. Are there intrauterine monitors that can give a woman some sense of whether labor is beginning to occur?
A. Correct.
Q. All right. And are those intrauterine monitors sometimes used to alert women to the possible signs of labor in an effort to prevent full-blown or full-fledged labor from occurring?
A. Not intrauterine monitors, no.
Q. Extrauterine monitors?
A. Extrauterine monitors.
Q. I used the wrong word. Are extrauterine monitors, then, used by the hospitals with which you're affiliated to assist in the prevention of premature delivery?
A. As a broad general statement, I think that would be true.
Q. All right. And is it your impression that again with respect to tocolytic treatment, that there is a range of outcome that one might expect from that intervention from no improvement to significant improvement?
A. Extrauterine monitoring is not an intervention. Tocolysis is.

Q. All right. Well, the use of that -- is it -- would you call it a treatment modality? What would you call it?
A. Diagnostic.
Q. All right. With the use of that diagnostic tool, has there been some data developed that show that that can reduce the occurrence of premature delivery?
A. Only if it's coupled with tocolysis, effective tocolysis.
Q. Dr. Speer, is there a benchmark figure which you have seen mentioned in the literature which respect to either low birth weight or prematurity that pertains to the author's conclusions that low birth weight and/or prematurity is associated with maternal smoking, in other words, an amount of smoking that is often referred to as the amount of smoking which produced the relationship or the statistical data that is set forth in the paper?
A. Could you simplify that question?
Q. Yeah. Is -- do -- do studies of maternal smoking which report relative risks for low birth weight or prematurity generally look at

that risk in women who smoke a pack a day or more?

A. I don't know the exact -- each study is somewhat different in the amount of smoking that has been studied. There does appear to be in some studies a relationship between the amount of smoking and the timing of smoking and subsequent outcome, as I summarized in my statement that I provided.

Q. All right. And in terms of the amount of smoking, if smoking is an -- is exerting an adverse pregnancy outcome, we would expect it to do so in accordance with the dose response principle, correct?

A. Depending upon the outcome would depend on whether that statement would be true.

Q. Can you think of any outcome that would be quote/unquote exempt from the operation of the dose response principle?

A. Not knowing everything about how tobacco works in causing these effects, it is difficult to formulate an answer to your question. However, it is well-known that in other models, you don't have to give very much of a substance to achieve an effect.

For example, if you give one dose of insulin, you are going to lower the blood sugar. You don't need a cumulative dose of insulin to lower the blood sugar. One dose will do it.

In other instances such as the closure of a patent ductus arteriosus with indomethacin, we have a dose response curve and it is related to numbers of doses and the ultimate level of the drug achieved in the bloodstream. Both of those are valid operational mechanisms for the effect of drugs on the human subject.

So in some instances, it appears that it is a dose response that maternal tobacco smoking confers on the fetus. In other instances, although I cannot point to them directly, it may well be a single exposure. I cannot say that that cannot occur.

Q. Well, it seems to me what you're differentiating are acute exposures and chronic exposures, correct?

A. Basically.

Q. All right. And for an cute or a chronic

exposure, the dose response principle would apply, correct?

A. Sometimes it takes less doses and sometimes it takes more doses.

Q. Right. Depending upon what we're talking about, is an acutely or a chronically mediated effect, correct?

A. Not totally.

Q. Are any of the effects that you are referring to in Exhibit 6 the result of non-chronic exposures?

A. Okay. In a hypothetical infant who already has uterine placental insufficiency who's exposed to a very high level of nicotine, there would be an acute single vaso -- could be an acute single vasoconstrictive event that would compromise a variety of organ systems, one of which could be the brain. Doesn't necessarily have to be a chronic exposure model in that sense.

Q. Do you have any idea what the circulating nicotine level would have to be in a fetus in order to affect the blood supply to the fetal brain?

A. It would undoubtedly depend on the

circumstances of the fetus. And, no, I do not know the exact bloodstream level that would have to be operational.

Q. Well, can you give us -- you qualified that by giving the qualifier "exact." Do you have any idea what the circulating level of nicotine would have to be in order to produce the effect that you've postulated?

A. No, I do not; which you didn't ask that originally.

Q. While we're on the subject of nicotine, briefly, are you familiar with the Doppler studies in human pregnancies that have looked at the effect of fetoplacental and uteroplacental circulation?

A. Only in a very general fashion. Once again, I would refer you to my obstetrical colleagues. I do not practice obstetrics.

Q. Do you know whether or not nicotine increases or decreases resistance in the fetoplacental circulation?

A. Not knowing exactly where in the fetoplacental circulation those studies addressed and not being familiar with the Doppler studies done in pregnant mothers and further not having

done those studies, I cannot answer your question.

Q. All right. How about in the uteroplacental vessels, do you know if nicotine increases or reduces resistance in the uteroplacental vessels?

A. If it causes vasoconstriction, it would cause increased resistance to flow. And if you're talking about the uterine artery, it is my understanding that nicotine does constrict flow.

Q. So a finding of reduced resistance in the uteroplacental vessels would be inconsistent with that, wouldn't it?

A. Would be inconsistent?

Q. Yes.

A. If you're -- remember, I'm talking about the uterine artery; you're talking about a whole blood circulating system when you say "uterine placental flow."

Q. Would those two things be consistent or inconsistent?

A. The way that we've defined them, inconsistent, because you're talking about a generality and I'm talking about a specific.

Q. The -- what is the -- what is the median weight of a baby at the beginning of the seventh month of pregnancy?
A. Are you defining --
Q. Excuse me, the end of the -- the end of the sixth month.
A. Are you talking about a 24-week fetus?
Q. Is that the median gestational age for a --
A. I go on weeks of gestation. I don't go on months. So I'm trying to find out what you're trying to ask me.
Q. Okay. Twenty-four weeks.
A. Twenty-four weeks? Again, I'd have to go back to the curves to give you a precise number, but probably about between 600 and 650 grams, right around there.
Q. Okay. And are you aware of any studies that have looked at the weight of 24-week-old fetuses in mothers who smoke versus mothers who don't smoke, in other words, what the weight difference is at that point of gestation?
A. I am unaware of any studies that have looked specifically at that particular gestation. However, I would anticipate that there is very

little, if any, fetal weight difference in those two.

Q. You would expect that difference to be exceedingly small, wouldn't you?

A. Yes.

Q. All right. And is that because most of fetal growth occurs in the seventh, eighth and ninth months?

A. Depends on your definition of "fetal growth." If you're talking about weight in grams, the answer is yes. If you're talking about cell multiplication, the answer is no.

Q. All right. Let's -- let's phrase it in terms of weight in grams.

A. Okay.

Q. All right. Does then the majority or the bulk of growth occur in the seventh, eighth and ninth months?

A. The daily increment of weight is higher in the end of pregnancy in actual grams than in the beginning of pregnancy. The ratio of growth weight to body weight may be equivalent.

Q. Well, for instance, in the first two trimesters, the fetus has grown to approximately 650 or so grams, correct?

A. Correct.
Q. And in the -- in the last trimester, the fetus is going to, in terms of median birth weight, grow an additional about 2,800 grams?
A. My point is, that 600-gram'er may well gain six grams to ten grams of weight a day whereas a 2,500-gram baby may grow 25 to 35 grams a day. So the amount of weight is proportional -- weight gain is proportional to the body weight.
Q. I see the point you're making.
A. And that stays relatively the same.
Q. I understand the point you're making. I'm asking about a slightly different one. Just in terms of the overall weight gain in the third trimester, would the mean overall weight gain be in the neighborhood of 2,850 grams? I mean, if we assumed is a -- does a mean weight of 3,500 grams sound about right to you?
A. 3,000 to 3,500.
Q. All right. And so we should subtract the 650 from that to determine the amount of weight gain that occurred in the third trimester?
A. Total amount of weight gained is larger in the last trimester than it is in the first

trimester --

Q. All right.

A. -- if you're just weighing in grams.

Q. All right. From the physiologic standpoint, would that not tell us that if smoking is going -- maternal smoking is going to mediate a significant -- or a change in birth weight, that the vast majority of that change is going to occur in the third trimester?

A. That seems to be the experience, correct.

Q. All right. And so would it be fair to say that if a woman quits smoking before the third trimester, the chances are, in terms of inter -- or low birth weight, the chances are that the birth weight of her baby is going to be essentially equivalent to that of a nonsmoker?

A. I'm not too sure it's the third trimester, but certainly if she stops somewhere after the first and the middle of the second, one would anticipate that that weight effect would disappear. And studies seem to have concluded that that is the case.

Q. All right. And do the studies also seem to indicate that with respect to any of the

health effects which you've testified about or that are in Exhibit 6, that if a woman quits her smoking by either the middle of the second trimester or the beginning of the third trimester, that she can reduce her risk to that of a nonsmoker?

A. She can reduce her risks in many areas. I think it will -- will have to be studied in further detail as to whether or not stopping smoking in the second trimester has an influence on the issue of mental retardation, because as I pointed out, cell division is the key during the first two trimesters of pregnancy. And the effect of tobacco may indeed be injurious to cell division and appropriate differentiation. And that may occur in the first trimester. At present, I don't think there is any data to say either way whether that is the case, but that certainly should be a concern.

Q. Putting mental retardation outside, then, of the ambit of the former question, would that be a true statement?

A. I think based on the knowledge we have at present, if the mother stops smoking in the

first trimester, early second trimester, many of the issues that you would -- inquired of me today would be ameliorated.

Q. And with respect to chronic exposures or any effects that are mediated through chronic exposures, would it be fair to say that the dose response principle applies?

A. Up to a degree. There is recent data in adults that I recently read that there appears -- this is a single study, certainly is not proving -- but there may well be a genetic trigger that is switched on after a certain amount of exposure to tobacco that even if the individual stops smoking, they are at significant increased risk to develop lung cancer at a later date because of the genetic switch. That type of phenomenon may also occur in babies. We don't know.

Q. That's pure speculation at this point?

A. That's speculation in babies. It looks like there is certainly something going on in adults. Beings how babies are replicating themselves at a much faster and higher rate and in a more complex manner than adults, I would be concerned.

Q. All right. But putting speculative issues aside, in terms of what we know, would it be fair to say that the chronic effects about which we know seem to behave in accordance with the dose response principle?
A. In many respects, yes.
Q. All right. And --
A. Oh, I remembered the name. Joel Dunnington. Joel Dunnington is the radiologist that asked me whether or not I would serve as an expert.
Q. All right. Do you know who --
A. I told you I would remember.
Q. Yeah. Do you know who -- do you know who Dr. Dunnington is?
A. As I said yesterday, I believe he's a radiologist at the M.D. Anderson Cancer Institute.
Q. Do you know anything else about him?
A. He's on the Harris County Medical Society Delegation to the Texas Medical Association.
Q. Do you have any --
A. He's an adult. He's overweight. He has a hip problem --
Q. Is --
A. -- or back, one or the other.

Q. Do you have any personal or professional relationship with him?
A. No.
Q. Do you know what Doctors Ought To Care is? Have you ever heard --
A. Yes.
Q. -- of that organization? Are you a member of it?
A. No.
Q. Do you know what it is?
A. Yes.
Q. What is it?
A. It's an organization founded by a physician named Alan Blum who is a family practitioner who in the group feels very strongly that tobacco is an undesirable product and uses what they term humor, although the tobacco industry doesn't appear to look at it as humor, to denigrate the product.
Q. What else do you know about Doctors Ought To Care, anything?
A. That's about it.
Q. All right. Do you know if Dr. Dunnington is the head of Doctors Ought To Care or is affiliated with it?

A. It wouldn't surprise me if Dr. Dunnington was a member, but I don't know for a fact.
Q. Has he made his anti-tobacco feelings clear to you?
A. He has been a relentless crusader against tobacco, one could phrase that probably correctly.
Q. All right. Have you ever read any of Dr. Dunnington's publications?
A. No.
Q. Do you know of any of his publications?
A. I would anticipate he is published, given the environment in which he practices.
Q. Did Dr. Dunnington tell you that he was going to be a witness in this case?
A. I have no earthly idea whether he's a witness or not.
Q. Have you ever heard Dr. Dunnington speak?
A. On what subject?
Q. Tobacco.
A. I've heard him say, you know, in various forums that -- you know, particularly in the TMA forum, that the TMA ought to come out very strongly against sales of tobacco to minors and also to regulate -- not -- "regulate" is

too strong a word -- to encourage strongly that tobacco smoking be eliminated within the TMA meetings. That was a number of years ago, and it has. And also coming out strongly that smoking in public places ought to be eliminated as a posture of the TMA. And I believe that is indeed TMA's posture.

MR. BLEVINS: Doctor, what time do you need to leave to get to your 12:00 o'clock?

THE WITNESS: We're fine.

MR. MINTON: I'm sorry?

THE WITNESS: Right around 12:00. And you can lock this room, if you wish.

MR. MINTON: Okay. There is a real possibility, I think, that we could finish up. I don't know what latitude you have in terms of being a few minutes late or something.

THE WITNESS: The latitude is a few minutes.

MR. MINTON: Okay.

THE WITNESS: Defined as less than five.

Q. (By Mr. Minton) Doctor, do you have children?
A. Two.
Q. How old are they?
A. Twenty-three and seventeen.
Q. Do either smoke?
A. No.
Q. Did you have a rule against smoking in your house?
A. No. They were the ones that in large measure were responsible, along with my wife, for my stopping.
Q. Have you -- have you familiarized yourself with any statistics regarding what percentage of women stop smoking when they learn they have become pregnant?
A. I can't give you an exact number. I know that a large -- well, at least I know a number do.
Q. And you were about to say large number but you caught yourself. It is a -- it is a number that you consider something relatively significant in terms of the clinical experience that you've had?
A. Correct.
Q. And are there another group of women who after first learning that they are pregnant quit

after their first prenatal visit because they are counseled to do so by their physician?

A. I would anticipate that is a true statement.

Q. All right. And in terms of the number of women who persist in smoking who don't spontaneously quit when they first learn they've become pregnant or quit as a result of advice of counseling from a physician, do you know what percentage of the women who were initially smokers that is?

A. No.

Q. It is your clinical experience, though, that there is a rather significant change in the -- in the percentage of women who smoke during pregnancy as opposed to women who were simply of child-bearing age?

A. I know that women who are smokers who become pregnant, there is a certain percentage of those women who will stop smoking either on the basis of knowledge that they are pregnant or upon counseling from the physician or other healthcare provider.

Q. Would it be fair to say, then, that a -- an estimate of women of child-bearing age would not be a reasonable estimate of women who

continue to smoke while they are pregnant?

A. I'm sorry?

Q. Would it be fair to say, then, that an estimate of the percentage of women of child-bearing age who smoke would not be an accurate estimate of the women who smoke while they are pregnant?

A. I think you probably have to define your population. Some populations will stop more readily than other populations.

Q. All right. And what is the basis of that belief?

A. As you pointed out, some people don't seek medical care and they don't necessarily have the educational background to understand the potential hazards of smoking. And thus if they don't have any knowledge, they have no basis to stop.

Q. All right. If one were to use general statistics, would the statement become -- you know, nationwide statistics, would the statement become a true one that the smoking prevalence among women of child-bearing age is not comparable to the smoking prevalence of women who are actually pregnant?

A. That may be. I don't know for a fact. I have no experience in looking at those particular data.
Q. Would it be fair to say that you don't have any data on the percentage of women who persist in smoking in pregnancy who are unaware of risks to their unborn children?
A. Correct.
Q. Would it be fair to say that it has been the mission of the -- of medical practitioners in the state of Texas to communicate that message to all women who are smokers and who are pregnant?
A. It is my understanding that that is something that the obstetrical community and other physicians try -- attempt to do, yes.
Q. All right. Now, you mentioned -- there is a reference in your disclosure document about women starting smoking while they are pregnant. Would it be fair to say that that's an extremely rare phenomenon?
A. I doubt it.
Q. Do you know -- have you seen any statistics on the number or percentage of women who begin to smoke while they are pregnant?

A. No. But if you're a teenager and that group has an unfortunate -- depending on the group you study, has an unfortunate ability to both smoke and get pregnant.

Q. If smoking -- if the chronically mediated effects of smoking adhere to the dose response principle, it is fair to say that as to those effects, there is a no-effect level, correct?

A. I followed your first phrase. I didn't get the last part.

Q. Are you familiar with the term "no-effect level"?

A. No.

Q. All right. I want you to assume that that means a level or a dose at which no symptom of any clinical significance is produced. Okay?

A. Okay.

Q. All right. If -- if the chronically mediated effects that are attributed to maternal smoking adhere to the dose response principle, there will be for each of those a no-effect level, correct?

A. You've made a rather large speculative leap of faith.

Q. Why is that?

A. It's an awfully big "if."
Q. What part of the "if"?
A. The whole part of the "if." You know, you're -- you're taking a dose response effect and then hypothesizing that in given individuals the given effect may or may not have no effect. And as I've already stated, we don't know that for sure. If you'd just stuck with the dose response, I would have been more than happy to agree with you.
Q. All right. Is it your opinion that there is a negative association between maternal smoking and congenital abnormalities of the newborn?
A. I don't think that that's been shown, at least shown in the totality of looking at the products of conception in smoking mothers and the incidence of chromosomal abnormalities throughout the extent of pregnancy.

If abortions are higher in mothers who smoke, which they appear to be, the most primary cause of abortion in the first trimester are chromosomal abnormalities. So if the abortions are higher, then you have a higher incidence of chromosomal abnormalities. In the totality of

pregnancy, because you have deaths that occurred during the pregnancy, you may have less chromosomal abnormalities in the surviving child.

Q. Do you know whether there has been --

A. So I don't know the information that you seek.

Q. All right. Is the first part of that speculation on your part in the sense that have you -- have you attempted to discern whether there were studies which looked at chromosomal abnormalities in abortuses to determine whether or not they existed and then correlated that with maternal smoking?

A. I'm not -- I don't have any information regarding the correlation of maternal smoking. However, I do -- am very aware that most abortions during the first trimester are the result of a variety of chromosomal abnormalities, triploidy and diploidy being two.

Q. All right. But whether -- whether abortions that some studies have attempted to statistically associate with maternal smoking, spontaneous abortions fall within that group, you don't know?

A. I don't know if the chromosomal analysis was done on those abortuses.
Q. All right. Now, putting that issue aside, is it fair to say that in terms of fetuses that live and -- that there is a negative association between maternal smoking and chromosomal abnormalities?
A. I don't know.
Q. Is there any association that you've ever heard of that's been linked between maternal smoking and congenital pneumonia?
A. How do you define "congenital pneumonia"?
Q. Is that an ICD-9 diagnostic entry?
A. It may well be and it would cover a variety of pathogens.
Q. All right. Have -- are you aware of any studies that have purported to link maternal smoking with congenital pneumonia?
A. Well, only by inference. And the inference is that if you take a population of patients that have premature or prolong rupture fetal membranes, that population has an increased risk of infection. One of the infectious diagnoses would be congenital pneumonia.
Q. How about meconium aspiration, are you aware

of any studies that link maternal smoking --

A. I think probably there are, because meconium passage in utero is related to fetuses who are small -- small-for-dates and pregnancies that have complications, particularly of the uterine placental unit.

So as a general statement, it would be anticipated that those pregnancies would have higher incidence of meconium passage and thus meconium aspiration syndrome as opposed to pregnancies without those problems.

Q. Are you aware of any studies that looked into the specific relationship between maternal smoking and meconium aspiration?

A. I have not looked for them, but it would not surprise me in the least that they exist.

Q. Okay. Well, regardless of whether it would surprise you, as you sit here today, you don't know whether they exist?

A. Correct.

Q. Is there a clinical diagnosis of large-for-date?

A. Sure.

Q. What is the birth weight --

A. Above the 90th percentile for gestational age.
Q. All right. Is that an adverse pregnancy outcome?
A. Can be, depending on the circumstances.
Q. What are the complications that are associated with that?
A. Depends on what it's due to. If it's due to infants of diabetic mothers, then you have an increased risk of hyaline membrane disease, you have an increased risk of what's termed birth trauma, because the baby is large and may be difficult to deliver from the -- from below via the vagina. They can have issues of hypoglycemia and hypocalcemia. Those are the primary problems of the infant of a diabetic mother.

If they happen to have a total -- let's see, transposition of the great vessels, those babies tend to be large. And obviously transposition of the great vessels is a rather serious congenital heart disease.

If they happen to have Becklet-Weidman Syndrome (phonetic), then they will have invalid seals and hypoglycemia and have a risk of increased mental retardation as

they grow older. Whether that retardation is due to the hypoglycemia or intrinsic to the condition is unknown.

Those are the -- and then you can have big babies because you have big parents, and those kids usually do fine except for the difficulty occasionally occasioned during the vaginal birth.

Q. So there is -- just as there is in the small-for-date classification, there -- in the large-for-date classifications there is large-normal and large-abnormal?

A. One could call it that.

Q. And in terms of the babies who are large-abnormal, are there significant costs that are associated with those adverse pregnancy outcomes as well?

A. Can be. Usually not as significant as prematures, but you certainly have costs associated.

MR. MINTON: Thank you, Dr. Speer.

THE WITNESS: You're welcome.

MR. BLEVINS: We'll reserve our questions until time of trial

THE VIDEOGRAPHER: The time is 12:06 p.m. We're off the record.

(Discussion off the record.)

THE VIDEOGRAPHER: The time is 12:07. We're on the record.

MR. MINTON: We wanted to put one more thing on the record, and that is the court reporter is going to just make a log of the depositions that were sent by Mr. Blevins to Dr. Speer so that we will have next to the deposition as Appendix "A" a list of the depositions that were provided to Dr. Speer so that we have a complete record of what he had. Did I get that right?

MR. BLEVINS: Yes.

THE WITNESS: Perhaps I should suggest not only depositions but any further documents that weren't entered as exhibits.

MR. MINTON: That are there on the cart?

THE WITNESS: Right. I don't know if -- I think they are all depositions, but in case they weren't, if

you'd like those pieces of paper, you now can have them.

THE VIDEOGRAPHER: The time is 12:08. We're off the record.

(Discussion off the record.)

MR. BLEVINS: Parties have reviewed the exhibits and find that Exhibit 11 is missing from those that will be attached to the deposition. Exhibit 11 was the drawing prepared by defense counsel in the review of issues related to Whites versus Blacks low birth weight babies, if I recall.

MR. MINTON: It was, yeah, distribution of birth weights. And it's evidently missing in action.

MR. BLEVINS: And the parties have agreed that the deposition may be completed without the attachment of that exhibit.

(Deposition concluded.)

CHANGE-CORRECTION PAGE

Please indicate changes on this sheet of paper, giving the page and line number, the change and the reason for the changes. Reason for changes are: (1) To clarify the record; (2) To conform to the facts; (3) To correct transcript errors.

PAGE	LINE	CORRECTION	REASON

SIGNATURE OF WITNESS

I have read the foregoing transcript of my deposition taken on the 4th day of September, 1997, and it is a true and accurate record of my testimony given at that time and place, except as to any corrections I have listed on the errata sheet(s).

_____
MICHAEL SPEER, M.D.

THE STATE OF TEXAS)

SUBSCRIBED AND SWORN TO BEFORE ME, the undersigned authority, on this the _____ day of _____, 1997.

_____
NOTARY PUBLIC IN AND FOR
THE STATE OF T E X A S

MY COMMISSION EXPIRES:
_____

IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
TEXARKANA DIVISION

THE STATE OF TEXAS	:	
	:	
Plaintiff	:	
	:	
VS.	:	
	:	CIVIL ACTION
THE AMERICAN TOBACCO COMPANY; R.J. REYNOLDS TOBACCO COMPANY; BROWN & WILLIAMSON TOBACCO CORPORATION; B.A.T. INDUSTRIES, P.L.C.; PHILIP MORRIS, INC.; LIGGETT GROUP, INC.; LORILLARD TOBACCO COMPANY, INC.; UNITED STATES TOBACCO COMPANY; HILL & KNOWLTON, INC.; THE COUNCIL FOR TOBACCO RESEARCH-USA, INC. (Successor to Tobacco Institute Research Committe); and THE TOBACCO INSTITUTE, INC.	:	NO. 5-96CV91 UNITED STATES JUDGE: DAVID FOLSOM UNITED STATES MAGISTRATE: WENDELL C. RADFORD

REPORTER'S CERTIFICATION
ORAL DEPOSITION OF MICHAEL SPEER
TAKEN SEPTEMBER 4, 1997

I, Linda Tate, Certified Shorthand Reporter in and for the State of Texas, hereby certify that this deposition transcript is a true record of the testimony given by the witness named herein, after said witness was duly sworn by me.

I further certify that I am neither attorney nor counsel for, related to, nor employed by any of the parties to the action in which this testimony was taken. Further, I am not a relative nor employee of any attorney of record in this cause, nor do I have a financial interest in this action.

Subscribed and sworn to on this the _____ day of _____, 1997.

_____
Linda Tate, CSR
CSR Certification No: 2965
Expiration Date: 12-31-97

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Houston, Texas 77010
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APPENDIX "A"

1) Transmittal letter dated August 8, 1997, transmitting the depositions and exhibits of Dr. Robert C. Woody, Dr. Robert Arrington, Dr. Robert J. Carpenter and Dr. Percy Luecke, Jr.

2) Deposition of Dr. Robert J. Carpenter, Pages 1 through 156, with index and condensed transcript and Exhibits 1 through 4 to Dr. Carpenter's deposition.

3) Deposition of Dr. Robert Arrington, Pages 1 through 172, with index and condensed transcript and Exhibits 1 and 2 to Dr. Robert Arrington's deposition.

4) Deposition of Dr. Percy E. Luecke, Pages 1 through 153, including index and condensed transcript and Exhibit No. 1 to Dr. Luecke's deposition.

5) Deposition of Dr. Robert C. Woody, Pages 1 through 178, including index and condensed transcript and Exhibits 1, 2 and 3 to Dr. Woody's deposition.

6) Transmittal letter dated August 17, 1997, enclosing the deposition of Dr. Benjamin Sachs.

7) Condensed Deposition of Dr. Benjamin Sachs taken in the Mississippi case, Pages 1 through 262.

8) Condensed deposition of Benjamin T. Sachs in the State of Florida versus American Tobacco Company case.

9) Condensed deposition of Dr. Jeane Ann McCarthy in the State of Florida versus American Tobacco.

10) Condensed version of the Robert Carpenter deposition in the Texas case.